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SINCE FILE ENTRY	TOTAL SESSION
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FILE COVERS 1907 - 12 Feb 2002 VOL 136 ISS 7  
FILE LAST UPDATED: 11 Feb 2002 (20020211/ED)

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The P indicator for Preparations was not generated for all of the CAS Registry Numbers that were added to the CAS files between 12/27/01 and 1/23/02. As of 1/23/02, the situation has been resolved. Searches and/or SDIs in the H/Z/CA/CAplus files incorporating CAS Registry Numbers with the P indicator executed between 12/27/01 and 1/23/02 may be incomplete. See the NEWS message on this topic for more information.

=> s adipocyte  
8852 ADIPOCYTE  
8255 ADIPOCYTES  
L1 11103 ADIPOCYTE  
(ADIPOCYTE OR ADIPOCYTES)

=> s fat cell  
147571 FAT  
75247 FATS  
183783 FAT  
(FAT OR FATS)  
1495791 CELL  
1345095 CELLS  
2030564 CELL  
(CELL OR CELLS)  
L2 3850 FAT CELL  
(FAT(W)CELL)

=> s l1 or l2  
L3 13281 L1 OR L2

=> s culture or cultured  
308181 CULTURE  
176332 CULTURES  
413016 CULTURE  
(CULTURE OR CULTURES)  
161001 CULTURED  
L4 505156 CULTURE OR CULTURED

=> s 13 and 14  
L5 1688 L3 AND L4

=> s protein or peptide  
1377294 PROTEIN  
892241 PROTEINS  
1585646 PROTEIN  
(PROTEIN OR PROTEINS)  
268030 PEPTIDE  
191197 PEPTIDES  
340098 PEPTIDE  
(PEPTIDE OR PEPTIDES)  
L6 1771287 PROTEIN OR PEPTIDE

=> s separat? or identi?  
263740 SEPARAT?  
228615 SEP  
12316 SEPS  
239831 SEP  
(SEP OR SEPS)  
399884 SEPD  
3 SEPDS  
399887 SEPD  
(SEPD OR SEPDS)  
73672 SEPG  
1 SEPGS  
73673 SEPG  
(SEPG OR SEPGS)  
447210 SEPN  
30290 SEPNS  
463072 SEPN  
(SEPN OR SEPNS)  
1143075 SEPARAT?  
(SEPARAT? OR SEP OR SEPD OR SEPG OR SEPN)  
994783 IDENTI?  
L7 2045360 SEPARAT? OR IDENTI?

=> s 15 and 16 and 17  
L8 154 L5 AND L6 AND L7

=> s preadipocyte  
1255 PREADIPOCYTE  
1031 PREADIPOCYTES  
L9 1525 PREADIPOCYTE  
(PREADIPOCYTE OR PREADIPOCYTES)

=> s 19 and 18  
L10 33 L9 AND L8

=> d ti 1-33

L10 ANSWER 1 OF 33 CAPLUS COPYRIGHT 2002 ACS

TI Induction of leptin expression in orbital **preadipocyte** fibroblasts

L10 ANSWER 2 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Insulin-responsive compartments containing GLUT4 in 3T3-L1 and CHO cells: regulation by amino acid concentrations

L10 ANSWER 3 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Altered expression of C/EBP family members results in decreased adipogenesis with aging

L10 ANSWER 4 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Fat depot origin affects fatty acid handling in cultured rat and human **preadipocytes**

L10 ANSWER 5 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Insulin/IGF-1 and TNF-.alpha. stimulate phosphorylation of IRS-1 at inhibitory Ser307 via distinct pathways

L10 ANSWER 6 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Autocrine regulation of human **preadipocyte** migration by plasminogen activator inhibitor-1

L10 ANSWER 7 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Growth and differentiation factor inhibitors and uses therefor

L10 ANSWER 8 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Inhibition of chicken **adipocyte** differentiation by in vitro exposure to monoclonal antibodies against embryonic chicken **adipocyte** plasma membranes

L10 ANSWER 9 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Priming with magnesium-deficient media inhibits **preadipocyte** differentiation via potential upregulation of tumor necrosis factor-.alpha.

L10 ANSWER 10 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Synthesis and secretion of plasminogen activator inhibitor-1 by human **preadipocytes**

L10 ANSWER 11 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI PPAR.gamma. ligand-dependent induction of STAT1, STAT5A, and STAT5B during adipogenesis

L10 ANSWER 12 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Molecular cloning of a major mRNA species in murine 3T3 **adipocyte** lineage. Differentiation-dependent expression, regulation, and identification as semicarbazide-sensitive amine oxidase

L10 ANSWER 13 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Angiotensin II receptors in human **preadipocytes**: role in cell cycle regulation

L10 ANSWER 14 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Understanding **adipocyte** differentiation

L10 ANSWER 15 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Regulation of triglyceride metabolism by PPARs: fibrates and thiazolidinediones have distinct effects

L10 ANSWER 16 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Differential expression of exons 1a and 1c in mRNAs for sterol regulatory element binding protein-1 in human and mouse organs and cultured cells

L10 ANSWER 17 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Regulation of the murine adipocyte fatty acid transporter gene by insulin

L10 ANSWER 18 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Differentiation-dependent expression of the brown adipocyte uncoupling protein gene: regulation by peroxisome proliferator-activated receptor  $\gamma$ .

L10 ANSWER 19 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Signal transduction pathway of acylation stimulating protein: involvement of protein kinase C

L10 ANSWER 20 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Cellular and molecular aspects of the regulation of adipogenesis

L10 ANSWER 21 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Cloning of a rat adipocyte membrane protein implicated in binding or transport of long-chain fatty acids that is induced during preadipocyte differentiation. Homology with human CD36

L10 ANSWER 22 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Differentiation of adipocyte precursors in a serum-free medium is influenced by glucocorticoids and endogenously produced insulin-like growth factor-I

L10 ANSWER 23 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Transiently and stably introduced CCAAT/enhancer-binding-protein genes are constitutively expressed in cultured cells

L10 ANSWER 24 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Identification of a fat cell enhancer: analysis of requirements for adipose tissue-specific gene expression

L10 ANSWER 25 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Analysis of a tissue-specific enhancer: ARF6 regulates adipogenic gene expression

L10 ANSWER 26 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Late expression of  $\alpha$ -2-adrenergic-mediated antilipolysis during differentiation of hamster preadipocytes

L10 ANSWER 27 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI A study of adipocyte precursor cells derived from brown adipose tissue: the expression of specific cell surface antigens during their differentiation in culture

L10 ANSWER 28 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Microtubule-associated protein 1A is the fibroblast HMW MAP undergoing mitogen-stimulated serine phosphorylation

L10 ANSWER 29 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Effects of cholera toxin on gene expression in brown preadipocytes differentiating in culture

L10 ANSWER 30 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Isolation and sequencing of a cDNA encoding the decarboxylase (E1).alpha. precursor of bovine branched-chain .alpha.-keto acid dehydrogenase complex. Expression of E1.α mRNA and subunit in maple-syrup-urine-disease and 3T3-L1 cells

L10 ANSWER 31 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Insulin stimulates glyceraldehyde-3-phosphate dehydrogenase gene expression through cis-acting DNA sequences

L10 ANSWER 32 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Cloning and expression of mouse fatty acid synthase and other specific mRNAs. Developmental and hormonal regulation in 3T3-L1 cells

L10 ANSWER 33 OF 33 CAPLUS COPYRIGHT 2002 ACS  
TI Hormone-sensitive lipase system and insulin stimulation of protein phosphatase activities in 3T3-L1 adipocytes

=> d bib ab 13 14 15 21 22 23 24 27

L10 ANSWER 13 OF 33 CAPLUS COPYRIGHT 2002 ACS  
AN 1999:11147 CAPLUS  
DN 130:149090  
TI Angiotensin II receptors in human preadipocytes: role in cell cycle regulation  
AU Crandall, David L.; Armellino, Douglas C.; Busler, Dennis E.; McHendry-Rinde, Barbara; Kral, John G.  
CS Wyeth-Ayerst Research, Princeton, NJ, 08543, USA  
SO Endocrinology (1999), 140(1), 154-158  
CODEN: ENDOAO; ISSN: 0013-7227  
PB Endocrine Society  
DT Journal  
LA English  
AB The role of angiotensin II (AII) in human preadipocyte physiol. has been investigated in primary cultures from human adipose tissue. Receptor binding studies indicated that human preadipocytes express a high affinity AII binding site of the AT1 subtype, as binding of <sup>125</sup>I-labeled [Sar1, Ile8]AII was rapid, saturable, and specific. As AII has previously been demonstrated to affect the cell cycle in adrenal and cardiac cells, the effect of AII on regulation of cycle progression was exmd. in human preadipocytes. Stimulation of preadipocytes with AII resulted in G1 phase progression of the cell cycle, as detd. by flow cytometric anal. AII treatment was assocd. with induction of expression of the mRNA for the cell cycle regulatory protein cyclin D1 in a dose-dependent manner. Pretreatment of cells with subtype-selective AT receptor ligands before AII stimulation indicated that the cyclin response was mediated via the AT1 receptor. The identity of the cells as preadipocyte was verified by culture in a defined differentiation medium, observing both leptin message expression and triglyceride accumulation by flow cytometry. These findings indicate that AII has early, receptor-mediated effects on cell cycle progression in human preadipocytes that may contribute to differentiation to the adipocyte phenotype.

RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 14 OF 33 CAPLUS COPYRIGHT 2002 ACS  
AN 1998:515549 CAPLUS  
DN 129:258060  
TI Understanding **adipocyte** differentiation  
AU Gregoire, Francine M.; Smas, Cynthia M.; Sul, Hei Sook  
CS Department of Nutritional Sciences, University of California, Berkeley,  
CA, USA  
SO *Physiol. Rev.* (1998), 78(3), 783-809  
CODEN: PHREA7; ISSN: 0031-9333  
PB American Physiological Society  
DT Journal; General Review  
LA English  
AB A review with 311 refs. The **adipocyte** plays a crit. role in energy balance. Adipose tissue growth involves an increase in **adipocyte** size and the formation of new **adipocytes** from precursor cells. For the last 20 yr, the cellular and mol. mechanisms of **adipocyte** differentiation have been extensively studied using **preadipocyte** culture systems. Committed **preadipocytes** undergo growth arrest and subsequent terminal differentiation into **adipocytes**. This is accompanied by a dramatic increase in expression of **adipocyte** genes including **adipocyte** fatty acid binding **protein** and lipid-metabolizing enzymes. Characterization of regulatory regions of adipose-specific genes has led to the **identification** of the transcription factors, peroxisome proliferator-activated receptor-.gamma. (PPAR-.gamma.) and CCAAT/enhancer binding **protein** (C/EBP), which play a key role in the complex transcriptional cascade during **adipocyte** differentiation. Growth and differentiation of **preadipocytes** is controlled by communication between individual cells or between cells and the extracellular environment. Various hormones and growth factors that affect **adipocyte** differentiation in a pos. or neg. manner have been **identified**. In addn., components involved in cell-cell or cell-matrix interactions such as **preadipocyte** factor-1 and extracellular matrix **proteins** are also pivotal in regulating the differentiation process. **Identification** of these mols. has yielded clues to the biochem. pathways that ultimately result in transcriptional activation  
via PPAR-.gamma. and C/EBP. Studies on the regulation of the these transcription factors and the mode of action of various agents that influence **adipocyte** differentiation will reveal the physiol. and pathophysiolog. mechanisms underlying adipose tissue development.

L10 ANSWER 15 OF 33 CAPLUS COPYRIGHT 2002 ACS  
AN 1997:431412 CAPLUS  
DN 127:104189  
TI Regulation of triglyceride metabolism by PPARs: fibrates and thiazolidinediones have distinct effects  
AU Auwerx, Johan; Schoonjans, Kristina; Fruchart, Jean-Charles; Staels, Bart  
CS Departement d'Atherosclerose, U. 325 INSERM, Institut Pasteur, Lille, 59019, Fr.  
SO *J. Atheroscler. Thromb.* (1996), 3(2), 81-89  
CODEN: JATHEH; ISSN: 1340-3478  
PB Japan Atherosclerosis Society  
DT Journal  
LA English  
AB The mol. mechanism by which hypolipidemic fibrates and antidiabetic thiazolidinediones exert their hypotriglyceridemic action are discussed. Increased activity of lipoprotein lipase (LPL), a key lipolytic enzyme,

and decreased levels of apolipoprotein C-III (apo C-III) seem to explain the hypotriglyceridemic effects of compds. Both fibrates and thiazolidinediones exert their action by activating transcription factors of the peroxisome proliferator-activated receptor (PPAR) family, thereby modulating the expression of the LPL and apo C-II genes. First, treatment

of rats with PPAR.alpha. activators, such as fibrates induced LPL mRNA and activity selectively in the liver. In contrast, the thiazolidinediones, which are high affinity ligands for PPAR.gamma., have no effect on liver, but induce LPL mRNA and activity levels in adipose tissue. In hepatocytes, fibrates, unlike the thiazolidinediones, induce LPL mRNA levels, whereas in preadipocyte cell lines the PPAR.gamma. ligand induces LPL mRNA levels much quicker and to a higher extent than fibrates. Second, apo C-III mRNA and protein prodn. strongly decrease in livers of fibrate- but not thiazolidinedione-treated animals. Fibrates also reduced apo C-III prodn. in primary cultures of rat and human hepatocytes. The modulation of the expression of the LPL and apo C-III genes by either PPAR.alpha. or .gamma. activators, correlates with the tissue-specific distribution of the resp. PPARs: PPAR.gamma. expression is restricted to adipose tissues, whereas PPAR.alpha. is expressed predominantly in liver. In both the LPL and apo C-III genes, sequence elements responsible for the modulation of their expression by activated PPARs have been identified which supports that the transcriptional regulation of these genes by fibrates and thiazolidinediones contributes significantly to their hypotriglyceridemic effects in vivo. Whereas thiazolidinediones pre-dominantly affect adipocyte LPL prodn. through activation of PPAR.gamma., fibrates exert their effects mainly in the liver via a PPAR.alpha.-mediated redn. in apo C-III prodn. This tissue-specific transcriptional regulation of genes involved in lipid metab. by PPAR activators and/or ligands might have important therapeutic implications.

L10 ANSWER 21 OF 33 CAPLUS COPYRIGHT 2002 ACS  
AN 1993:514293 CAPLUS  
DN 119:114293  
TI Cloning of a rat adipocyte membrane protein implicated in binding or transport of long-chain fatty acids that is induced during preadipocyte differentiation. Homology with human CD36  
AU Abumrad, Nada A.; El-Maghrabi, M. Raafat; Lopez, Ellen; Amri, Ez Zoubir; Grimaldi, Paul A.  
CS Dep. Physiol. Biophys., State Univ. New York, Stony Brook, NY, 11794, USA  
SO J. Biol. Chem. (1993), 268(24), 17665-8  
CODEN: JBCHA3; ISSN: 0021-9258  
DT Journal  
LA English  
AB A cDNA for an adipocyte membrane protein, implicated in the transport of long-chain fatty acids, was isolated by screening with a synthetic oligonucleotide derived from the N-terminal sequence of the protein. The 88-kDa adipocyte membrane protein was previously identified by covalent labeling with N-sulfosuccinimidyl esters of long-chain fatty acids which irreversibly inhibited fatty acid transport by 75%. The cDNA (FAT, 2432 base pairs (bp)) contained 70 bp of 5'-untranslated sequence, an open reading frame encoding a 472-amino acid protein with a predicted mol. mass of 52,466, and 940 bp of 3'-untranslated sequence with 2 polyadenylation signal sequences but with no polyadenylation tail. The deduced protein sequence predicted 2 transmembrane segments and 10 potential N-linked glycosylation sites. Extensive glycosylation most

likely explains why the mol. mass of the isolated **protein** (88 kDa) is different from that deduced from the cDNA sequence (53 kDa). The sequence of FAT is 85% homologous with that of glycoprotein IV (CD36) identified in human platelets and in lactating mammary epithelium. Consistent with this, a polyclonal antibody against CD36 reacted with **adipocyte** plasma membranes and detected a single band at 88 kDa. Northern blot anal. of RNA obtained from rat adipose tissue and probed with the cDNA identified 2 major transcripts of 4.8 and 2.9 kilobases which were abundant in heart, intestine, fat, muscle, and testis. The mRNAs were not detectable in **cultured** adipose cell lines (Ob1771, 3T3F442A) at the fibroblastic stage but was strongly induced during the differentiation process and by treatment of **preadipocytes** with dexamethasone, conditions that were also assocd. with an increase in oleate transport. In contrast, the fibroblastic cell lines 3T3-C2 and L929, which do not differentiate, did not express the mRNAs at all stages of **culture**. The data suggest that FAT and CD36 belong to a family of **proteins** that bind/transport long-chain fatty acids or function as regulators of these processes.

L10 ANSWER 22 OF 33 CAPLUS COPYRIGHT 2002 ACS  
AN 1993:421117 CAPLUS  
DN 119:21117  
TI Differentiation of **adipocyte** precursors in a serum-free medium is influenced by glucocorticoids and endogenously produced insulin-like growth factor-I  
AU Nougues, Jean; Reyne, Yves; Barenton, Bruno; Chery, Therese; Garandel, Veronique; Soriano, Josette  
CS Unite Differ. Cell. Croissance, Inst. Natl. Rech. Agron., Montpellier, 34060, Fr.  
SO Int. J. Obes. (1993), 17(3), 159-67  
CODEN: IJOBDP; ISSN: 0307-0565  
DT Journal  
LA English  
AB Stromal vascular cells from rabbit perirenal adipose tissue differentiated at a high frequency in a chem.-defined serum-free medium contg. insulin, transferrin, T3, and dexamethasone. The omission from the **culture** medium of dexamethasone resulted in a lack of adipose conversion. Addn. of IGF-I increased glycerol-3-phosphate dehydrogenase (GPDH) activity. The conditioned media from **adipocyte** precursor cells contained measurable quantities of immunoreactive IGF-I as detd. by RIA after neutralization of IGF binding **proteins** interference. Dexamethasone increased IGF-I secretion during the first 7 days after plating and decreased IGF-I binding to conditioned media. Three mol. forms of IGF binding **proteins** (IGFBPs) were identified by Western ligand blots in conditioned media, with Mr = 40,000, 29,000 and 25,000. The major form (Mr = 29,000) was decreased by dexamethasone. In contrast, the Mr = 24,000 form was increased. Specific binding of <sup>125</sup>I-labeled IGF-I to rabbit **adipocyte** precursor cells was more effectively inhibited by unlabeled IGF-I than by unlabeled IGF-II or insulin. The electrophoretic migration of crosslinked <sup>125</sup>I-IGF-I to microsomal membranes revealed a complex with Mr = 130,000 under reducing conditions corresponding to the .alpha.-subunit of the IGF-I receptor. The addn. of IGF-I monoclonal antibody to rabbit **adipocyte** precursor cells **cultured** in serum-free medium inhibited [<sup>3</sup>H]thymidine incorporation and decreased (50%) GPDH specific activities. This inhibitory effect was overcome by the addn. of exogenous IGF-I. Thus, stromal vascular cells isolated from perirenal adipose tissue

secrete IGF-I and IGFBPs, possess IGF-I receptors and respond to exogenous and endogenous IGF-I. In addn., the results indicate that the IGF-I autocrine/paracrine role in replication and differentiation of rabbit adipocyte precursors is influenced by glucocorticoids.

L10 ANSWER 23 OF 33 CAPLUS COPYRIGHT 2002 ACS  
AN 1992:585987 CAPLUS  
DN 117:185987  
TI Transiently and stably introduced CCAAT/enhancer-binding-protein genes are constitutively expressed in cultured cells  
AU Xanthopoulos, Kleanthis G.; Cannon, Paul D.; Robinson, Gregory S.; Mirkovitch, Jovan; Darnell, James E., Jr.  
CS Cent. Biotechnol., Karolinska Inst., Huddinge, S-141 57, Swed.  
SO Eur. J. Biochem. (1992), 208(2), 501-9  
CODEN: EJBCAI; ISSN: 0014-2956  
DT Journal  
LA English  
AB CCAAT/enhancer-binding protein (C/EBP) is expressed in certain cell types including hepatocytes and adipocytes. To understand the mechanisms that control the expression of the mouse C/EBP gene in the liver as well as in adipocytes, the authors studied both the endogenous gene and transfected C/EBP gene constructs. The initiation site of transcription was identified and a strong liver-specific DNase-I hypersensitive site located at -3 kb, which does not contribute functionally to the regulation of the gene in a variety of either transiently or stably transfected cells with constructs which include sequences 1toreq.6-kb upstream of the transcription start. C/EBP gene expression during the transition from preadipocytes to adipocytes was controlled at the level of transcription. However, adipocytes stably transfected with constructs that include -3.3 kb upstream of the C/EBP gene do not express the reporter genes in a differentiation-specific manner. Several DNA-binding proteins were detected that interact with the upstream sites of the C/EBP gene. Those include 2 labile and 2 heat-stable site-specific DNA-binding proteins that are present in nuclear exts. from several tissues and cultured cell lines.

L10 ANSWER 24 OF 33 CAPLUS COPYRIGHT 2002 ACS  
AN 1992:525443 CAPLUS  
DN 117:125443  
TI Identification of a fat cell enhancer: analysis of requirements for adipose tissue-specific gene expression  
AU Graves, Reed A.; Tontonoz, Peter; Platt, Kenneth A.; Ross, Susan R.; Spiegelman, Bruce M.  
CS Dana-Farber Cancer Inst., Boston, MA, 02115, USA  
SO J. Cell. Biochem. (1992), 49(3), 219-24  
CODEN: JCEBD5; ISSN: 0730-2312  
DT Journal; General Review  
LA English  
AB A review with 20 refs. of the authors recent results on the identification and characterization of a far upstream enhancer from the murine aP2 gene that directs high levels of adipose-specific gene expression in transgenic mice. Although the proximal promoter contg. AP-1 and C/EBP binding sites is capable of directing differentiation-dependent gene expression in cultured adipocytes, these constructs are essentially inactive in the tissues of transgenic mice. -5.4 Kb of the 5'-flanking region were required to direct heterologous

gene (chloramphenicol acetyl transferase; CAT) expression to the adipose tissue of transgenic mice. Deletion anal. was used to **identify** a 520 bp enhancer at -5.4 kb of the ap2 gene that can direct high levels of gene expression specifically to the adipose tissue of transgenic mice. This enhancer also functions in a differentiation-dependent manner in **cultured adipocytes** and cannot be transactivated in **preadipocytes** by C/EBP. Mol. anal. indicates that several **cis**- and **trans**- acting acting elements, though not C/EBP, contribute to the specificity and potency of this enhancer. The potential uses of this enhancer to target the expression of **proteins** to the adipose depot in transgenic animals in discussed.

L10 ANSWER 27 OF 33 CAPLUS COPYRIGHT 2002 ACS  
 AN 1991:79277 CAPLUS  
 DN 114:79277  
 TI A study of **adipocyte** precursor cells derived from brown adipose tissue: the expression of specific cell surface antigens during their differentiation in **culture**  
 AU Lee, S. R.; Cryer, A.  
 CS Coll. Cardiff, Univ. Wales, Cardiff, CF1 1ST, UK  
 SO J. Dev. Physiol. (1990), 13(2), 105-13  
 CODEN: JDPHDH; ISSN: 0141-9846  
 DT Journal  
 LA English  
 AB Using cell specific anti-**adipocyte** sera and an immuno-pptn. procedure, the nature of the cell surface antigens characterizing **adipocytes** from rat brown adipose tissue was investigated. Initially the ability of anti-sera, raised against adipose plasma membrane preps. of white or brown adipose tissue, to distinguish between membrane preps. derived from either tissue was confirmed. Anal. of the plasma membranes derived from brown adipose and similar preps. labeled with 125I revealed the presence of specific externally disposed mature brown **adipocyte**-specific antigens. The specifically immunoptd. antigens had mol. wts. of 70,000, 56,000 and 23,000. None of these antigens were cross immunoptd. by antisera to mature white **adipocyte** membranes. The presence of the brown adipose-specific antigens on the surface of differentiating **adipocyte** precursor cells derived from rat brown adipose tissue was demonstrated using a labeled-second antibody cellular immunoassay. The expression of the immunoreactivity assocd. with these antigens was shown to be an early event in the differentiation program of the cells in vitro. The functional **identity** and possible roles of these antigens in the control of brown **adipocyte** differentiation now becomes accessible to further exptl. investigation.

=> file biosis

COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
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FULL ESTIMATED COST

47.30 47.45

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

	SINCE FILE ENTRY	TOTAL SESSION
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FILE COVERS 1969 TO DATE.  
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT  
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 6 February 2002 (20020206/ED)

The BIOSIS file has been reloaded. Enter HELP RLOAD and HELP REINDEXING  
for details.

=> s 110

739 PREADIPOCYTE  
1093 PREADIPOCYTES  
1462 PREADIPOCYTE  
    (PREADIPOCYTE OR PREADIPOCYTES)  
6955 ADIPOCYTE  
9195 ADIPOCYTES  
12860 ADIPOCYTE  
    (ADIPOCYTE OR ADIPOCYTES)  
115080 FAT  
15692 FATS  
125687 FAT  
    (FAT OR FATS)  
2079521 CELL  
1551926 CELLS  
2688187 CELL  
    (CELL OR CELLS)  
4459 FAT CELL  
    (FAT(W) CELL)  
336253 CULTURE  
182412 CULTURES  
453160 CULTURE  
    (CULTURE OR CULTURES)  
182636 CULTURED  
3 CULTUREDS  
182638 CULTURED  
    (CULTURED OR CULTUREDS)  
1180371 PROTEIN  
442697 PROTEINS  
1362663 PROTEIN  
    (PROTEIN OR PROTEINS)  
208624 PEPTIDE  
104949 PEPTIDES  
266194 PEPTIDE  
    (PEPTIDE OR PEPTIDES)  
316515 SEPARAT?  
791696 IDENTI?  
L11       26 L9 AND L8

=> s 111 not 126

L26 NOT FOUND

The L-number entered could not be found. To see the definition  
of L-numbers, enter DISPLAY HISTORY at an arrow prompt (=>).

=> s 111 not 110  
739 PREADIPOCYTE  
1093 PREADIPOCYTES  
1462 PREADIPOCYTE  
    (PREADIPOCYTE OR PREADIPOCYTES)  
6955 ADIPOCYTE

9195 ADIPOCYTES  
12860 ADIPOCYTE  
(ADIPOCYTE OR ADIPOCYTES)  
115080 FAT  
15692 FATS  
125687 FAT  
(FAT OR FATS)  
2079521 CELL  
1551926 CELLS  
2688187 CELL  
(CELL OR CELLS)  
4459 FAT CELL  
(FAT (W) CELL)  
336253 CULTURE  
182412 CULTURES  
453160 CULTURE  
(CULTURE OR CULTURES)  
182636 CULTURED  
3 CULTUREDS  
182638 CULTURED  
(CULTURED OR CULTUREDS)  
1180371 PROTEIN  
442697 PROTEINS  
1362663 PROTEIN  
(PROTEIN OR PROTEINS)  
208624 PEPTIDE  
104949 PEPTIDES  
266194 PEPTIDE  
(PEPTIDE OR PEPTIDES)  
316515 SEPARAT?  
791696 IDENTI?  
L12 0 L11 NOT L10  
  
=> d ti l11  
  
L11 ANSWER 1 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI Adipose depot-specific expression of cIAP2 in human **preadipocytes** and modulation of expression by serum factors and TNFalpha.  
  
=> d ti l11 1-26  
  
L11 ANSWER 1 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI Adipose depot-specific expression of cIAP2 in human **preadipocytes** and modulation of expression by serum factors and TNFalpha.  
  
L11 ANSWER 2 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI Altered expression of C/EBP family members results in decreased adipogenesis with aging.  
  
L11 ANSWER 3 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI Fat depot origin affects fatty acid handling in **cultured** rat and human **preadipocytes**.  
  
L11 ANSWER 4 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI Autocrine regulation of human **preadipocyte** migration by plasminogen activator inhibitor-1.  
  
L11 ANSWER 5 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI Metallothionein gene expression and secretion in white adipose tissue.

L11 ANSWER 6 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI Adipogenesis in thyroid eye disease.

L11 ANSWER 7 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI A branched DNA signal amplification assay to quantitate messenger RNA of human uncoupling **proteins** 1, 2, and 3.

L11 ANSWER 8 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI Inhibition of chicken **adipocyte** differentiation by in vitro exposure to monoclonal antibodies against embryonic chicken **adipocyte** plasma membranes.

L11 ANSWER 9 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI Priming with magnesium-deficient media inhibits **preadipocyte** differentiation via potential upregulation of tumor necrosis factor-alpha.

L11 ANSWER 10 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI Synthesis and secretion of plasminogen activator inhibitor-1 by human **preadipocytes**.

L11 ANSWER 11 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI PPARgamma ligand-dependent induction of STAT1, STAT5A, and STAT5B during adipogenesis.

L11 ANSWER 12 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI Angiotensin II receptors in human **preadipocytes**: Role in cell cycle regulation.

L11 ANSWER 13 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI Adipose tissue extracellular matrix: Newly organized by **adipocytes** during differentiation.

L11 ANSWER 14 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI Differential expression of exons 1a and 1c in mRNAs for sterol regulatory element binding **protein**-1 in human and mouse organs and **cultured** cells.

L11 ANSWER 15 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI Regulation of the murine **adipocyte** fatty acid transporter gene by insulin.

L11 ANSWER 16 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI Differentiation-dependent expression of the brown **adipocyte** uncoupling **protein** gene: Regulation by peroxisome proliferator-activated receptor gamma.

L11 ANSWER 17 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI Signal transduction pathway of acylation stimulating **protein**: Involvement of **protein** kinase C.

L11 ANSWER 18 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI Cloning of a rat **adipocyte** membrane **protein** implicated in binding or transport of long-chain fatty acids that is induced during **preadipocyte** differentiation: Homology with human CD36.

L11 ANSWER 19 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI TRANSIENTLY AND STABLY INTRODUCED CCAAT-ENHANCER-BINDING-**PROTEIN** GENES ARE CONSTITUTIVELY EXPRESSED IN **CULTURED** CELLS.

L11 ANSWER 20 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI LATE EXPRESSION OF ALPHA-2-ADRENERGIC-MEDIATED ANTI-LIPOLYSIS DURING  
DIFFERENTIATION OF HAMSTER **PREADIPOCYTES**.

L11 ANSWER 21 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI CYTOPLASMIC PROTEINS OF PORCINE **ADIPOCYTES**  
IDENTIFICATION WITH MONOCLONAL ANTIBODIES.

L11 ANSWER 22 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI EFFECTS OF CHOLERA TOXIN ON GENE EXPRESSION IN BROWN **PREADIPOCYTES**  
DIFFERENTIATING IN CULTURE.

L11 ANSWER 23 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI S-100 PROTEIN IN WHITE **PREADIPOCYTES** AN IMMUNOELECTRON  
MICROSCOPIC STUDY.

L11 ANSWER 24 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI ISOLATION AND SEQUENCING OF A COMPLEMENTARY DNA ENCODING THE  
DECARBOXYLASE  
E-1-ALPHA PRECURSOR OF BOVINE BRANCHED-CHAIN ALPHA KETO ACID  
DEHYDROGENASE  
COMPLEX EXPRESSION OF E-1-ALPHA MESSENGER RNA AND SUBUNIT IN MAPLE-SYRUP  
URINE DISEASE AND 3T3-L1 CELLS.

L11 ANSWER 25 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI LIPO PROTEIN LIPASE EC-3.1.1.34 CONTENT IN OB-17  
**PREADIPOCYTES** DURING ADIPOSE CONVERSION IMMUNO FLUORESCENT  
LOCALIZATION OF THE ENZYME.

L11 ANSWER 26 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
TI DEVELOPMENT OF LIPO PROTEIN LIPASE ACTIVITY AND ACCUMULATION OF  
TRI ACYL GLYCEROL IN DIFFERENTIATING 3T3-L-1 **ADIPOCYTES** EFFECTS  
OF PROSTAGLANDIN F-2-ALPHA 1 METHYL-3-ISOBUTYL XANTHINE PROLACTIN AND  
INSULIN.

=> d bib ab 7 12 13 19

L12 HAS NO ANSWERS

L1	11103	SEA FILE=CAPLUS ABB=ON	PLU=ON	ADIPOCYTE
L2	3850	SEA FILE=CAPLUS ABB=ON	PLU=ON	FAT CELL
L3	13281	SEA FILE=CAPLUS ABB=ON	PLU=ON	L1 OR L2
L4	505156	SEA FILE=CAPLUS ABB=ON	PLU=ON	CULTURE OR CULTURED
L5	1688	SEA FILE=CAPLUS ABB=ON	PLU=ON	L3 AND L4
L6	1771287	SEA FILE=CAPLUS ABB=ON	PLU=ON	PROTEIN OR PEPTIDE
L7	2045360	SEA FILE=CAPLUS ABB=ON	PLU=ON	SEPARAT? OR IDENTI?
L8	154	SEA FILE=CAPLUS ABB=ON	PLU=ON	L5 AND L6 AND L7
L9	1525	SEA FILE=CAPLUS ABB=ON	PLU=ON	PREADIPOCYTE
L10	33	SEA FILE=CAPLUS ABB=ON	PLU=ON	L9 AND L8
L11	26	SEA FILE=BIOSIS ABB=ON	PLU=ON	L9 AND L8
L12	0	SEA FILE=BIOSIS ABB=ON	PLU=ON	L11 NOT L10

=> d bib ab l11 7 12 13 19

L11 ANSWER 7 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
AN 2000:372285 BIOSIS  
DN PREV200000372285  
TI A branched DNA signal amplification assay to quantitate messenger RNA of  
human uncoupling **proteins** 1, 2, and 3.

AU Zhou, Lubing (1); Cryan, Ellen V.; Minor, Lisa K.; Gunnet, Joseph W.; Demarest, Keith T.

CS (1) Endocrine Therapeutics, Drug Discovery, R.W. Johnson Pharmaceutical Research Institute, 1000 Route 202, Raritan, NJ, 08869 USA

SO Analytical Biochemistry, (June 15, 2000) Vol. 282, No. 1, pp. 46-53.  
print.  
ISSN: 0003-2697.

DT Article

LA English

SL English

AB Uncoupling **proteins** (UCP) are inner mitochondrial membrane transporters which dissipate the proton gradient, releasing stored energy as heat. Three subtypes of UCP have been **identified** so far. The regulation of UCP expression is mainly controlled at the transcriptional level, thus making the measurement of UCP mRNA beneficial for both diagnosis and research of weight disorders and diabetes. We have developed  
an assay using the branched DNA signal amplification assay (bDNA assay)  
to quantitatively measure the mRNA levels for human UCP1, 2, and 3. UCP-subtype-specific primers were designed for the assay. RNA transcripts of each UCP generated by in vitro transcription were used to validate the specificity and sensitivity of the assay. The quantitative measurement of UCP mRNA was further demonstrated with **cultured** cells and human tissue. A comprehensive survey of UCP expression from 17 human tissues measured by the newly developed assay is provided. The method described here offers a rapid, sensitive, specific, and quantitative assay for measurement of human UCP mRNA.

L11 ANSWER 12 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

AN 1999:74774 BIOSIS

DN PREV199900074774

TI Angiotensin II receptors in human **preadipocytes**: Role in cell cycle regulation.

AU Crandall, David L. (1); Armellino, Douglas C.; Busler, Dennis E.; McHendry-Rinde, Barbara; Kral, John G.

CS (1) Wyeth-Ayerst Res., CN 8000, Princeton, NJ 08543 USA

SO Endocrinology, (Jan., 1999) Vol. 140, No. 1, pp. 154-158.  
ISSN: 0013-7227.

DT Article

LA English

AB The role of angiotensin II (AII) in human **preadipocyte** physiology has been investigated in primary **cultures** from human adipose tissue. Receptor binding studies indicated that human **preadipocytes** express a high affinity AII binding site of the AT1 subtype, as binding of 125I-labeled (Sar1,Ile8)AII was rapid, saturable, and specific. As AII has previously been demonstrated to affect the cell cycle in adrenal and cardiac cells, the effect of AII on regulation of cycle progression was examined in human **preadipocytes**. Stimulation of **preadipocytes** with AII resulted in G1 phase progression of the cell cycle, as determined by flow cytometric analysis. AII treatment was associated with induction of expression of the messenger RNA for the cell cycle regulatory **protein** cyclin D1 in a dose-dependent manner. Pretreatment of cells with subtype-selective AT receptor ligands before AII stimulation indicated that the cyclin response was mediated via the AT1 receptor. The **identity** of the cells as **preadipocyte** was verified by **culture** in a defined differentiation medium, observing both leptin message expression and

triglyceride accumulation by flow cytometry. These findings indicate that AII has early, receptor-mediated effects on cell cycle progression in human **preadipocytes** that may contribute to differentiation to the **adipocyte** phenotype.

L11 ANSWER 13 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
AN 1998:475614 BIOSIS  
DN PREV199800475614  
TI Adipose tissue extracellular matrix: Newly organized by **adipocytes** during differentiation.  
AU Nakajima, Ikuyo (1); Yamaguchi, Takahiro; Ozutsumi, Kyouhei; Aso, Hisashi  
CS (1) Cell. Biol. Lab., Dep. Anim. Physiol., Natl. Inst. Anim. Industry,  
Nordindanchi, Tsukuba, Ibaraki 305 Japan  
SO Differentiation, (Aug., 1998) Vol. 63, No. 4, pp. 193-200.  
ISSN: 0301-4681.  
DT Article  
LA English  
AB The distribution of eight types of extracellular matrix (ECM) **proteins** (type I-VI) collagen, laminin and fibronectin) in the skeletal muscle of Japanese Black cattle was determined by indirect immunofluorescence using specific antibodies against each **protein**. ECM **proteins** were well organized in the intramuscular connective tissue: type I, II, III collagen and fibronectin were localized primarily in the perimysium, type V and VI collagen in both the perimysium and endomysium, and type IV collagen and laminin were virtually confined to the endomysium. In the loose connective tissue holding the **adipocytes** together to form a tissue mass between the muscular bundles, seven of the ECM **proteins** not type II collagen were relatively abundant in a disordered arrangement. Further analysis by *in vitro* immunocytochemical staining also demonstrated that a stromal-vascular **preadipocyte** cell line (BIP cell), derived from Japanese Black cattle, synthesized various ECMs in much the same way as fibroblasts. Exponentially growing BIP cells with a fibroblastic phenotype were found to produce type II, V, and VI collagens, in addition to the other previously identified connective tissue glycoproteins of mouse 3T3 **preadipocytes**. When confluent **preadipocyte** cultures were stimulated with adipogenic medium, a fibrillar network of ECM was observed to bridge the intercellular space and connect adjacent cell surfaces. During **adipocyte** differentiation, type III collagen and laminin were arranged in a non-fibrous structure, and type II collagen was only barely detected. These results are supported by the staining of the adipose tissue, where all ECM **proteins** studied except type II collagen were stained intensely. These data indicate that *in vivo* under conditions permissive for adipose conversion, the production and organization of ECM, accompanied by hyperplasia and hypertrophy of precursor cells, gives rise to adipose tissue in skeletal muscle with its own ECM products. These data further suggest that each ECM **protein** might have some role for the **adipocytes** in forming tissue.

L11 ANSWER 19 OF 26 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
AN 1992:501281 BIOSIS  
DN BA94:119806  
TI TRANSIENTLY AND STABLY INTRODUCED CCAAT-ENHANCER-BINDING-PROTEIN GENES ARE CONSTITUTIVELY EXPRESSED IN CULTURED CELLS.  
AU XANTHOPOULOS K G; CANNON P D; ROBINSON G S; MIRKOVITCH J; DARNELL J E JR  
CS KAROLINSKA INSTITUTE, CENTER BIOTECHNOLOGY, NOVUM, S-141 57 HUDDINGE,

SWED.  
SO EUR J BIOCHEM, (1992) 208 (2), 501-509.  
CODEN: EJBCAI. ISSN: 0014-2956.  
FS BA; OLD  
LA English  
AB CCAAT/enhancer-binding protein (C/EBP) is expressed in certain cell types including hepatocytes and adipocytes. In order to understand the mechanisms that control the expression of the mouse C/EBP gene in the liver as well as in adipocytes, we have studied both the endogenous gene and transfected C/EBP gene constructs. The initiation site of transcription was identified and a strong liver-specific DNase-I hypersensitive site located at -3 kb, which does not appear to contribute functionally to the regulation of the gene in a variety of either transiently or stably transfected cells with constructs which include sequence up to 6-kb upstream of the transcription start. C/EBP gene expression during the transition from preadipocytes to adipocytes was shown to be controlled at the level of transcription. However, adipocytes stably transfected with constructs that include -3.3 kb upstream of the C/EBP gene do not express the reporter genes in a differentiation-specific manner. We detected several DNA-binding proteins that interact with the upstream sites of the C/EBP gene. Those include two labile and two heat-stable site-specific DNA-binding proteins that are present in nuclear extracts from several tissues and cultured cell lines.

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